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A Walsh–Fourier Analysis of the Effects of Moderate Maternal Alcohol Consumption on Neonatal Sleep-State Cycling

DAVID S. STOFFER, MARK S. SCHER, GALE A. RICHARDSON, NANCY L. DAY,
and PATRICIA A. COBLE*

Recent discussions of the functional significance of ultradian rhythms emphasize their importance to human physiology. Over the past 25 years, electroencephalographic (EEG) sleep patterns have been used in assessing the cerebral and central nervous system maturation of neonates. Through an interdisciplinary effort, spectral (Fourier) methods have been developed to discriminate between the various stages of sleep based on EEG recordings. Nevertheless, there has been little effort to develop methods for the statistical analysis of sleep-state cycling. In particular, attention has primarily been focused on the ultradian rhythm of sleep as it cycles between two states, active or rapid eye movement (REM) and quiet or non-REM sleep. There are, however, several components of REM and non-REM sleep, as well as a transitional state (indeterminate sleep) and abrupt alterations of state (arousal). Moreover, few studies have investigated the effects of prenatal alcohol exposure on the neurophysiological development of infants as assessed through sleep or EEG patterns. In this article the theory of Walsh–Fourier analysis for discrete-valued (categorical) time series is used to investigate the spectral components of EEG sleep-state patterns of infants whose mothers abstained from drinking during pregnancy, and infants of mothers who used moderate amounts of alcohol continuously during pregnancy. The sample for this study is part of a larger cohort of women participating in a longitudinal study of substance use during pregnancy. The analysis is based on the finite Walsh–Fourier transform that is defined in terms of the Walsh functions. The square-wave Walsh functions form a complete orthonormal sequence on $[0, 1)$ and take on only two values, $+1$ and -1 (“on” and “off”). This allows correlation of per-minute infant sleep-state records with square waveforms, and analysis of sleep-state cycles relative to per-minute switches from one sleep stage to another (termed sequency). The analysis consists of two parts. First, the article considers the problem of detecting whether a common sleep pattern exists in the unexposed and exposed groups of neonates separately. As expected, it is concluded that a common signal does exist in each group. Next, the spectral components of the sleep-state signals of the two groups are compared, and the differences between the two groups are discussed.

KEY WORDS: Analysis of variance (power); Categorical time series; Neonatal EEG sleep patterns; Prenatal alcohol exposure; Sequency domain; Spectral analysis.

1. INTRODUCTION

Electroencephalographic (EEG) sleep patterns have been used to assess cerebral maturation and neurophysiological organization of the developing central nervous system in premature, full-term, and older infants (Dreyfus-Brisac 1964, 1968, 1970, 1979; Ellingson and Peters 1980; Parmelee et al. 1967, 1968; Prechtl, Aliyama, Zinkin, and Grant 1968). From such studies, abnormal EEG sleep patterns have been associated with specific clinical conditions identified in either the prenatal or perinatal periods. Also, EEG sleep abnormalities can predict medical and behavioral difficulties in neonates and infants in the absence of clinical abnormalities (Crowell, Kapuniai, Boychuk, Light, and Hodgman 1982; Lombroso 1982; Tharp, Cukier, and

Monod 1981; Thoman, Denenberg, Sievel, Zeidner, and Becker 1980).

In the past, there has been considerable research on the effects on infant outcome of moderate and excessive maternal alcohol use during pregnancy. Few studies, however, have monitored neurophysiological measures of sleep cycling and arousal as independent biological measures to clinical and/or neurobehavioral assessments. Optimally, prognostic assessments of the neonate and infant require serial EEG sleep recordings. From these recordings, careful consideration should be given to the lability of state and the disruption of the expected rapid eye movement (REM) and non-REM components of the neonatal or infant sleep cycle.

The sample for this study is part of a larger cohort of infants born to women participating in a longitudinal study of moderate substance use during pregnancy. Women attending the prenatal clinic at a large, urban, university-affiliated obstetrical hospital were asked to participate in the study. A detailed description of the study design and the methods used for measuring alcohol use has been reported elsewhere (Day, Wagener, and Taylor 1985; Scher, Richardson, Coble, Day, and Stoffer, 1988).

From a total of 780 deliveries, 55 newborns (27 males

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and 28 females) were selected for EEG sleep studies. Overall, the mothers of these infants were representative of the larger study cohort of women. We limit the discussion of EEG sleep findings to the first neonatal recording of these infants obtained at 24 to 36 hours after birth. These two-hour (approximately) EEG sleep recordings were routinely obtained on swaddled infants approximately 45 minutes to 1 hour after a morning feeding. Recordings for male infants were obtained prior to circumcision. All neonatal EEG sleep recordings were conducted in a quiet, environmentally controlled room designated for neonatal and infant sleep studies.

Neonatal sleep EEG's were recorded with a Nihon Kohden (model 4200) 21-channel polygraph. Sixteen channels were devoted to EEG's and five channels recorded additional physiologic measures; electrode placement for the newborn recordings followed the standard international 10/20 system, with modification recommended for neonates. Throughout the recording period, behavioral observations were continuously noted on the recording paper by the EEG technician. Such observations included eyes open, eyes closed, eye movements, body movements, jerks and twitches with indication of the body part(s) involved, startles, sucking, vocalizations, respiratory irregularities, and the infant appearing quiet. Environmental noises were noted to differentiate evoked from spontaneous arousals.

The neonatal EEG sleep recordings were scored independently by an electroencephalographer who was not aware of the prenatal substance exposure of these infants. Recordings were scored for EEG state, REM's, arousals, and body movements, using one-minute scoring epochs. Scoring was based on operational definitions with representative neonatal and infant EEG recording samples of each area. The development of the scoring manual incorporated existing standard definitions for awake, active, and quiet sleep states in the neonate, and for REM and the various non-REM sleep stages in older infants. Additional sleep behavior, including indeterminate or transitional sleep, arousals, and phasic REM activity have been included in the scoring manual. Full scoring manuals are available from Scher on written request.

Infant sleep state was categorized (per minute) into six possible states:

1. Quiet Sleep—Trace Alternant
2. Quiet Sleep—High Voltage
3. Indeterminate Sleep
4. Active Sleep—Low Voltage
5. Active Sleep—Mixed
6. Awake.

We assess the effects of regular (albeit moderate) alcohol consumption during pregnancy on infant sleep-state cycling. In particular, our goal is to compare unexposed infants to infants who were continuously exposed to alcohol. Of the 55 infants selected for sleep studies, 12 had mothers who abstained from drinking alcohol throughout their pregnancy, and 12 had mothers who used alcohol moderately and consistently throughout their pregnancy.

The remaining cases involved women who consumed alcohol occasionally, with no regularity, throughout their pregnancy; typically, these women consumed 0–1 drink per month during the second and third trimesters [see Scher et al. (1988) for a description of the drinking habits and demographics of the 55 selected women]. Hence, in our analysis infants are categorized into two groups: the first group contains 12 infants whose mothers abstained from drinking alcohol during pregnancy, and the second group contains 12 infants whose mothers consumed alcohol regularly and at an average rate of at least three drinks per week throughout pregnancy. The actual sleep-state records (to 120 minutes) for infants in group 1 and group 2 are plotted in Figures 1 and 2, respectively, using the aforementioned sleep-state labels, 1–6.

Although a considerable amount of analysis has been devoted to using EEG series to discriminate among sleep stages (e.g., see Gersch, Martinelli, Yonemoto, Lew, and McEwan 1979), investigations of sleep-state cycling typically only result in empirical descriptions about cycling behavior. Such descriptions deal in generalities and focus mainly on the slower cycles that can be detected by visual examination of sleep-state records. Although specific sleep segments can follow predictable state changes between active (REM) and quiet (non-REM) sleep, statistical models and methodologies are essential for both systematic detection of the common cyclic components of normal sleep and detection of alterations or disruptions in the physiological rhythms of sleep state.

The analysis of infant sleep state is accomplished via spectral methods using the Walsh–Fourier transform. This enables us to analyze the sleep-state cycles (which we may think of as square waveforms) in terms of square waves and sequency (switches per unit time). The advantage of this method over trigonometric (Fourier) methods was empirically demonstrated by Beauchamp (1975). In particular, Beauchamp concluded that the respective roles of Walsh and Fourier spectral analyses are clear: “Where the signal is derived from sinusoidally-based waveforms . . . then Fourier analysis is relevant. Where the signal contains sharp discontinuities and a limited number of levels . . . then Walsh analysis is appropriate” (p. 103).

Our analysis consists of two parts. First, we consider the problem of detecting whether a common signal (sleep pattern) exists among the 12 records in each group. Next, we consider the problem of detecting the differences, if any, between the common signals of the abstainers (group 1) and the moderate alcohol users (group 2). That is, we are interested in whether there is an exposure effect.

In Section 2 we briefly discuss existing Walsh–Fourier theory. In Section 3 we present the statistical models and methodology used in our analysis, and in Section 4 we present and discuss the analysis results.

2. WALSH–FOURIER ANALYSIS

In this section we give a brief account of the existing Walsh–Fourier theory for stationary time series. Specific

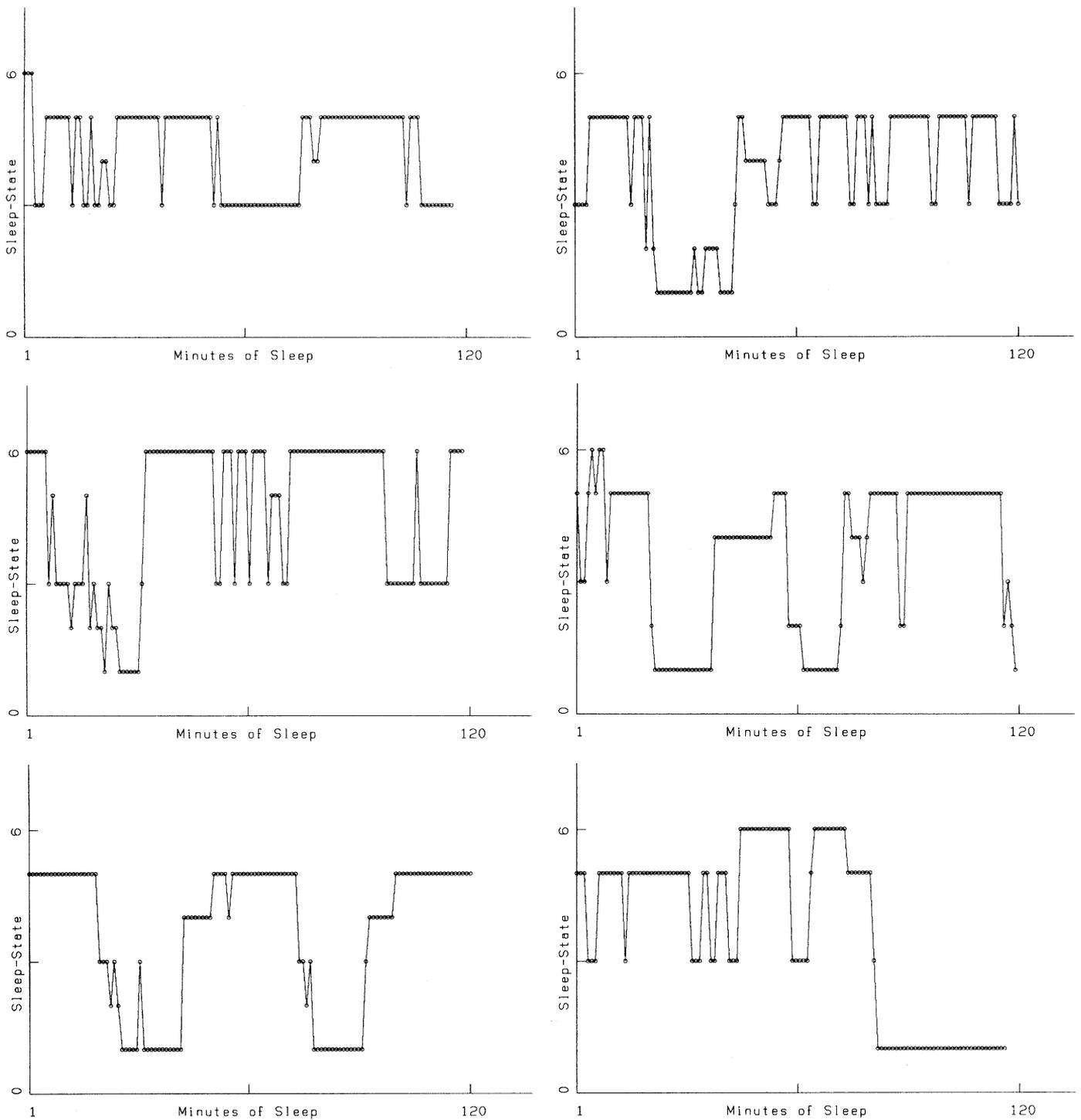


Figure 1. Sleep-State Records of Infants Whose Mothers Abstained From Drinking During Pregnancy (group 1).

details and references may be found in Kohn (1980a,b), Morettin (1981), and Stoffer (1987).

The Walsh functions form a complete orthonormal sequence on $[0, 1)$ and take on only two values, +1 and -1 ("on" and "off"). They are ordered by the number of zero-crossings (or switches), called *sequency*. Let $W(n, \lambda)$ ($n = 0, 1, 2, \dots$; $0 \leq \lambda < 1$) denote the n th sequency-ordered Walsh function; then, $W(n, \cdot)$ makes n zero-crossings in $[0, 1)$. The first eight discrete, sequency-ordered Walsh functions, $W(n, m/N)$ ($m, n = 0, 1, \dots, 7$), corresponding to a sample of length $N = 2^3$ are shown as

the rows (columns) of the Walsh-ordered Hadamard matrix, $\mathbf{H}_w(3)$:

$$\mathbf{H}_w(3) = \begin{bmatrix} 1 & 1 & 1 & 1 & 1 & 1 & 1 & 1 \\ 1 & 1 & 1 & 1 & -1 & -1 & -1 & -1 \\ 1 & 1 & -1 & -1 & -1 & -1 & 1 & 1 \\ 1 & 1 & -1 & -1 & 1 & 1 & -1 & -1 \\ 1 & -1 & -1 & 1 & 1 & -1 & -1 & 1 \\ 1 & -1 & -1 & 1 & -1 & 1 & 1 & -1 \\ 1 & -1 & 1 & -1 & -1 & 1 & -1 & 1 \\ 1 & -1 & 1 & -1 & 1 & -1 & 1 & -1 \end{bmatrix}$$

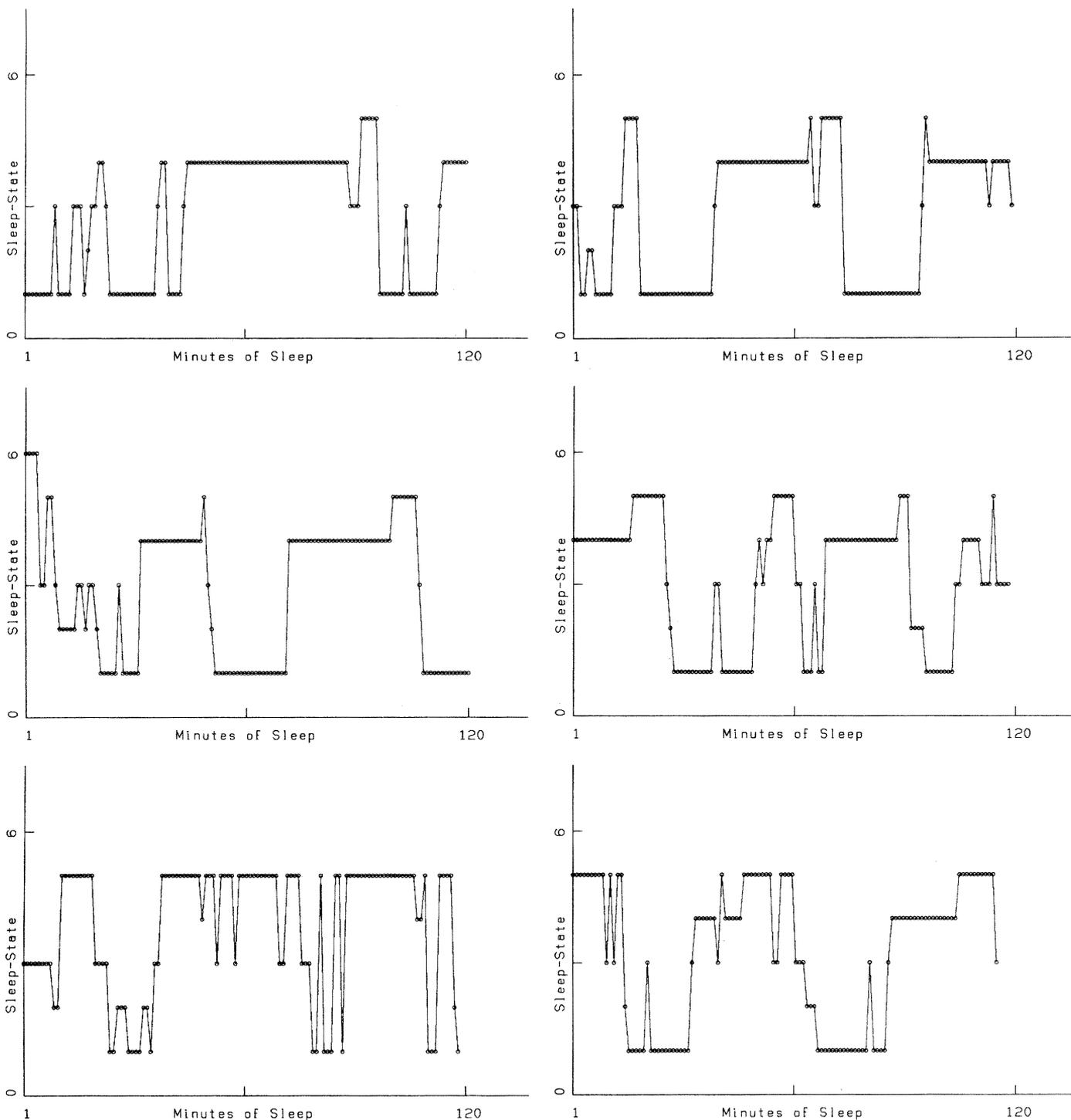


Figure 1 (continued).

Let $X(0), X(1), \dots, X(N - 1)$ be a sample of length $N = 2^p$ ($p > 0$ integer) from stationary time series $\{X(n), n = 0, \pm 1, \pm 2, \dots\}$, with absolutely summable autocovariance function $\gamma(h) = \text{cov}\{X(n), X(n + h)\}$ ($h = 0, \pm 1, \pm 2, \dots$). We assume for now that the constant mean value of $X(n)$ is 0. Let $W(n, \lambda)$ be the n th Walsh function in sequency order, and let

$$d_N(\lambda) = N^{-1/2} \sum_{n=0}^{N-1} X(n)W(n, \lambda), \quad 0 \leq \lambda < 1, \quad (2.1)$$

be the finite (or discrete) Walsh–Fourier transform of the data. The logical covariance of $X(n)$ (see Kohn 1980a) is defined as $\tau(j) = N^{-1} \sum_{k=0}^{N-1} \gamma(j \oplus k - k)$, where by $j \oplus k$ we mean the dyadic addition of j and k . It can then be shown that the variance of $d_N(\lambda)$ is given by

$$\text{var}\{d_N(\lambda)\} = \sum_{j=0}^{N-1} \tau(j)W(j, \lambda). \quad (2.2)$$

Taking the limit ($N \rightarrow \infty$) in (2.2), we have that $\text{var}\{d_N(\lambda)\}$

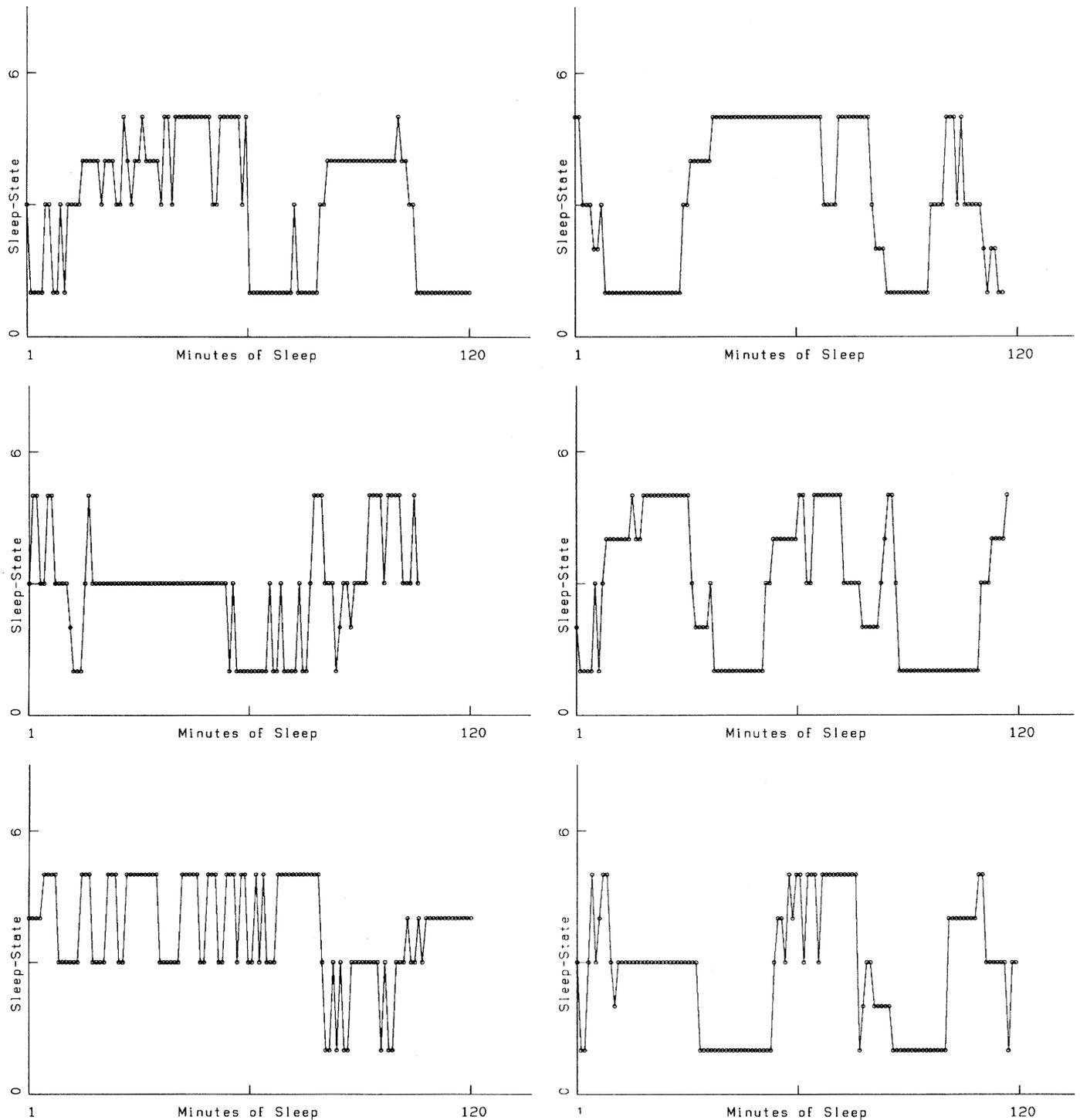


Figure 2. Sleep-State Records of Infants Whose Mothers Used Alcohol During Pregnancy (group 2).

→ $f(\lambda)$, where

$$f(\lambda) = \sum_{j=0}^{\infty} \tau(j)W(j, \lambda), \quad 0 \leq \lambda < 1, \quad (2.3)$$

is called the Walsh-Fourier spectral density of $X(n)$. We note that $f(\lambda)$ exists, since the absolute summability of $\gamma(h)$ implies the absolute summability of $\tau(j)$. See Kohn (1980a) for details.

If $X(0), X(1), \dots, X(N - 1)$ is a sample of length $N = 2^p$, the finite transform (2.1) is calculated for $\lambda_N =$

m/N ($m = 0, 1, \dots, N - 1$). Since the discrete Walsh functions are symmetric in their arguments for $N = 2^p$, that is, $W(n, m/N) = W(m, n/N)$ ($m, n = 0, 1, \dots, N - 1$), the value λ_N in the finite Walsh-Fourier transform corresponds to sequency. As with the usual Fourier analysis, if the mean of the series is unknown, the only sequency of the form $\lambda_N = m/N$ for which the transform cannot be evaluated is at the 0 ($m = 0$) sequency (see Stoffer 1987).

For samples of length $N = 2^p$ there exist fast methods

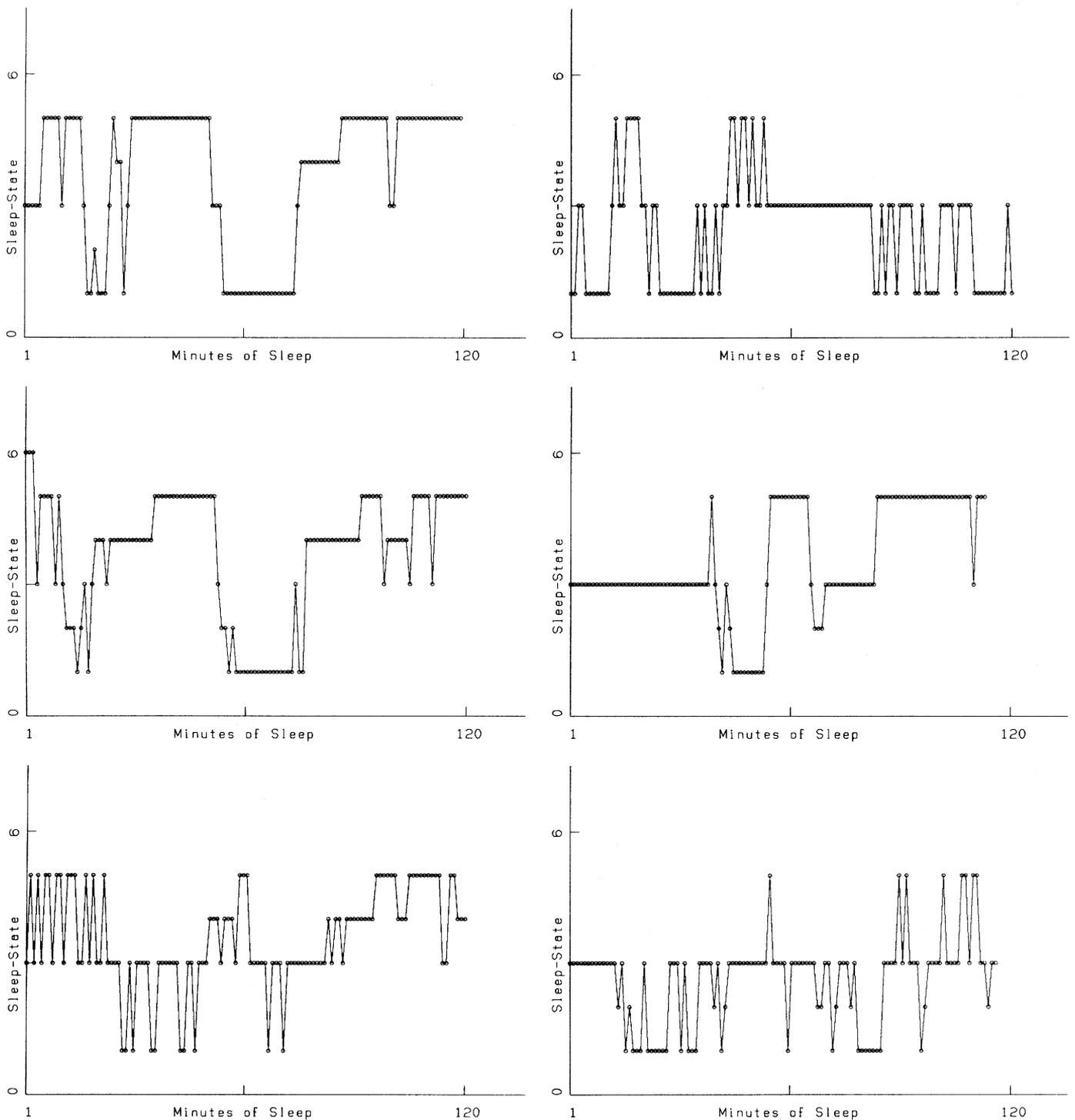


Figure 2 (continued)

for computing the finite Walsh-Fourier transform. See Ahmed and Rao (1975, chap. 6), who also gave a FORTRAN computer subroutine, for details.

Various asymptotic results relating the convergence ($N \rightarrow \infty$) of $d_N(\lambda_N)$ to $f(\lambda)$ exist. Many of these results, especially those that are applicable to categorical time series, were discussed in Stoffer (1987). Other references include Kohn (1980a,b), Morettin (1981, 1983), and Stoffer (1985). Of particular interest to us is the fact that under appropriate conditions (such as the existence of higher mo-

ments), $d_N(\lambda_N)$ converges in law to a normal variate with mean 0 and variance $f(\lambda)$, given by (2.3). Also, we mention that unlike the usual Fourier analysis, the asymptotic covariance of the Walsh-Fourier transform at two distinct frequencies is not necessarily 0.

Finally, we focus on the stationarity assumption. Clearly, one needs stationarity (or at least stationary variation about a time-varying process mean) to formally define a spectrum; however, as noted by Brillinger and Tukey (1984), "stationarity is not a necessity for useful spectrum analysis.

... Apparent lack of stationarity in the data is *not* a reason to give up either the idea of a spectrum, or the hope of useful results in estimating (averages over) that spectrum. It is a reason to be careful about computational practice (else leakage may bury the information that might have helped you)" (pp. 1084–1085).

3. STATISTICAL MODELS AND METHODOLOGY

First we address the problem of detecting whether a common signal exists in each group. Let $X_{ql}(n)$ denote the sleep state of infant q ($q = 1, \dots, 12$) in group l ($l = 1, 2$) at minute n ($n = 0, 1, \dots, N - 1$), and suppose that $EX_{ql}(n) = \theta_{ql}$, for all n . Let $S_l(n)$ be a mean-0, stationary, discrete-valued signal with Walsh–Fourier spectrum $f_{S_l}(\lambda)$. We hypothesize that $S_1(n)$ [$S_2(n)$] is common to all infant sleep-state series in group 1 [group 2]. Let $\varepsilon_{ql}(n)$ be independent realizations of a mean-0 stationary, discrete-valued process with common Walsh–Fourier spectrum $f_\varepsilon(\lambda)$ such that $S_1(n)$ [$S_2(n)$] and $\varepsilon_{q1}(n)$ [$\varepsilon_{q2}(n)$] are uncorrelated (but not necessarily independent), and $\varepsilon_{i1}(n)$ and $\varepsilon_{j2}(n)$ are mutually independent ($i, j = 1, \dots, 12$). Following Stoffer (1987), we propose the following model as a realistic decomposition of the sleep-state processes:

$$X_{ql}(n) = \theta_{ql} + S_l(n) + \varepsilon_{ql}(n) \quad (3.1)$$

($q = 1, \dots, 12; l = 1, 2; n = 0, 1, \dots, N - 1$).

Let $d_{N,ql}(\lambda_{m,N})$ denote the Walsh–Fourier transform of $X_{ql}(n)$ at sequency $\lambda_{m,N} = m/N$ ($q = 1, \dots, 12; l = 1, 2; m = 1, \dots, N - 1$). Then, under appropriate conditions, for fixed $\lambda_{m,N}$ the transform of (3.1) has the following representation:

$$d_{N,ql}(\lambda_{m,N}) = U_{lm} + Z_{qlm} + 0_{a.s.}(1), \quad (3.2)$$

where for each m , U_{lm} are independent $N(0, f_{S_l}(\lambda_{m,N}))$ variates, Z_{qlm} are independent $N(0, f_\varepsilon(\lambda_{m,N}))$ variates independent of U_{lm} , and $0_{a.s.}(1)$ is a variate that tends to 0, almost surely, as $N \rightarrow \infty$. See Stoffer (1987) for details.

Our analysis is based on the representation (3.2). The problem of detecting whether there is a common signal in each group is accomplished by testing the null hypotheses $H_1: S_1(n) = 0$ (all n), and $H_2: S_2(n) = 0$ (all n), or equivalently, $H_1: f_{S_1}(\lambda) = 0$ ($0 < \lambda < 1$), and $H_2: f_{S_2}(\lambda) = 0$ ($0 < \lambda < 1$), respectively.

Let

$$d_{N,l}^2(\lambda_{m,N}) = [(12)^{-1} \sum_{q=1}^{12} d_{N,ql}(\lambda_{m,N})]^2, \quad l = 1, 2, \quad (3.3)$$

and define appropriate sums of squares

$$\sum_{q=1}^{12} [d_{N,ql}(\lambda_{m,N}) - d_{N,l}(\lambda_{m,N})]^2, \quad l = 1, 2. \quad (3.4)$$

We note that in view of (3.2), (3.3) and (3.4) have the almost sure ($N \rightarrow \infty$) representations $[f_{S_l}(\lambda_{m,N}) + (12)^{-1}f_\varepsilon(\lambda_{m,N})]\chi_{11}^2$ ($l = 1, 2$) and $f_\varepsilon(\lambda_{m,N})\chi_{11}^2$, respectively, where the χ_v^2 variates are independent (χ_v^2 denotes a chi-squared variate with v df).

The test of the null hypotheses H_1 and H_2 may be examined at particular sequencies by comparing

$$\frac{12 d_{N,l}^2(\lambda_{m,N})}{\sum_{q=1}^{12} [d_{N,ql}(\lambda_{m,N}) - d_{N,l}(\lambda_{m,N})]^2/11}, \quad l = 1, 2, \quad (3.5)$$

with an $F_{1,11}$ distribution, respectively.

Next, we address the problem of detecting whether there are differences between the signals $S_1(n)$ and $S_2(n)$ for groups 1 and 2, respectively. Here we suppose that $S_l(n)$ in (3.1) may be decomposed as $S_l(n) = \mu(n) + \alpha_l(n)$ ($l = 1, 2$), where $\alpha_l(n)$ may be viewed as the sleep signal that is particular to group l and is uncorrelated with $\mu(n)$, which represents a signal common to all sleep-state records. We further suppose that $\alpha_1(n)$ and $\alpha_2(n)$ are independent processes with common Walsh–Fourier spectrum $f_S(\lambda)$ and $\mu(n)$ has Walsh–Fourier spectrum $f_\mu(\lambda)$, so that $f_{S_l}(\lambda) = f_\mu(\lambda) + f_S(\lambda)$ ($l = 1, 2$). In this case (3.2) may be written as

$$d_{N,ql}(\lambda_{m,N}) = U_m + V_{lm} + Z_{qlm} + 0_{a.s.}(1), \quad (3.6)$$

where for each m , the U 's, V 's, and Z 's are mutually independent $N(0, f_\mu(\lambda_{m,N}))$, $N(0, f_S(\lambda_{m,N}))$, and $N(0, f_\varepsilon(\lambda_{m,N}))$ variates, respectively. The comparison of the group signals is accomplished by testing the null hypothesis $H_3: \alpha_1(n) = \alpha_2(n) = 0$ (all n), or equivalently, $H_3: f_S(\lambda) = 0$ ($0 < \lambda < 1$).

Define $d_{N,\cdot}(\lambda_{m,N}) = (2)^{-1} \sum_{l=1}^2 d_{N,l}(\lambda_{m,N})$, where $d_{N,l}(\lambda_{m,N})$ is defined in (3.3), and appropriate sums of squares

$$\sum_{l=1}^2 [d_{N,l}(\lambda_{m,N}) - d_{N,\cdot}(\lambda_{m,N})]^2 \quad (3.7)$$

and

$$\sum_{l=1}^2 \sum_{q=1}^{12} [d_{N,ql}(\lambda_{m,N}) - d_{N,l}(\lambda_{m,N})]^2. \quad (3.8)$$

In view of (3.6), (3.7) and (3.8) have the almost sure ($N \rightarrow \infty$) representations $[f_S(\lambda_{m,N}) + (12)^{-1}f_\varepsilon(\lambda_{m,N})]\chi_1^2$ and $f_\varepsilon(\lambda_{m,N})\chi_{22}^2$, respectively, where the χ_v^2 variates are independent.

The test of the null hypothesis $H_3: f_S(\lambda) = 0$ ($0 < \lambda < 1$) can be examined at particular sequencies by comparing

$$\frac{12 \sum_{l=1}^2 [d_{N,l}(\lambda_{m,N}) - d_{N,\cdot}(\lambda_{m,N})]^2}{\sum_{l=1}^2 \sum_{q=1}^{12} [d_{N,ql}(\lambda_{m,N}) - d_{N,l}(\lambda_{m,N})]^2/22} \quad (3.9)$$

with an $F_{1,22}$ distribution.

We must emphasize the fact that the tests given in (3.5) and (3.9) are appropriate at a single a priori determined sequency. Since these tests are to be used for an exploratory analysis, special consideration must be given to the overall error rate. This problem was discussed in the context of trigonometric (Fourier) analysis by Brillinger (1980) and Shumway (1988, p. 70). Brillinger stated that one might want to set down overall test statistics such as $\sup_m F(\lambda_{m,N})$, $\sum_m F(\lambda_{m,N})$, and $\prod_m [1 + (N - 1)F(\lambda_{m,N})]$, where $F(\lambda_{m,N})$ denotes the F statistic (3.5) or (3.9). But

he claims: “It seems however that the use of such test statistics provides too brutal a summary of the data collected” (pp. 245–246). Shumway suggested that the way to overcome the problem of the overall error rate involved in making simultaneous statements about the value of the spectrum over more than one frequency is to use Bonferroni’s inequality. In this context, a bound on the overall error rate is $\sum_{m=1}^K \alpha_m$, where α_m is the null significance of the test (3.5) or (3.9) at sequency $\lambda_{m,N}$, and K is the number of sequencies under consideration. Thus, in the investigation of a common group sleep pattern, we use levels of .001 and .01 to identify the strong and moderate peak periods. Subsequently, in the between-group analysis we will be able to focus on a few particular sequencies of interest; in this case, we use a level of .01 to indicate group differences. Nevertheless, we do feel that since this is an exploratory analysis, it is appropriate for our presentation to indicate crossings of the level-.05 threshold.

To use the fast Walsh–Fourier transform given by Ahmed and Rao (1975), the standard procedure of padding the data to a length that is a power of 2 was employed. In particular, the actual sample lengths of the data ran anywhere from 113 to 127 minutes. Thus the spectral components were computed by extending each series to $128 = 2^7$ minutes by padding each sleep-state record with zeros.

4. RESULTS AND DISCUSSION

First, we discuss the results of the tests for a common signal in each of the two groups. Figure 3 shows the log of the average periodogram (3.3) for group 1 (abstainers) and group 2 (users). Although the two periodograms are similar in many respects, there is typically more power at the common peak periods for group 1 than for group 2. Figure 4 shows a plot of the statistic (3.5) for group 1 and group 2 at all sequencies except the 0 sequency and indicates the null significances of .01 and .001. To facilitate the examination of the spectral components, Table 1 lists the values of the statistic (3.5) for each group at selected sequencies. From these results we conclude that a common sleep-state signal exists in each group, although the evidence of this for group 2 is not overwhelming. Moreover, the analyses indicate a broad range of peak periods; these peak periods are listed in Table 1, along with their null significance. Note that in the context of the sequency domain, a peak period of p minutes means one switch every p minutes, as opposed to the usual trigonometric definition of one cycle every p minutes.

Since we concluded that there is a common signal in each of the two groups, we address the problem of whether there are any differences between the common signals of each group. Figure 5 shows a plot of the statistic (3.9) for all sequencies, except 0, and indicates the null significances of .05 and .01. The values of the statistic (3.9) at selected sequencies are also listed in Table 1. These results indicate strong differences between the group signals at periods of 4.41, 2.33, and 1.38 minutes ($m = 29, 55,$ and $93,$ respectively, in Table 1) and differences at periods of 5.12,

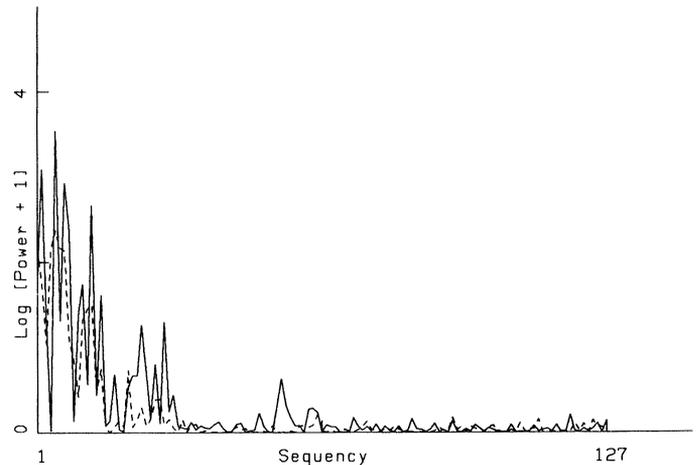


Figure 3. Average Periodogram (3.3) for Group 1 (—) and Group 2 (---).

1.08, and 1.03 minutes ($m = 25, 119,$ and $124,$ respectively, in Table 1). In particular, we note that, with the exception of the 1.03 minute period, these peak periods are present in the abstainer group (group 1) and absent in the user group (group 2). We also note that these differences are essentially at the “fast” periods—those ranging between approximately one and five minutes. From the results listed in Table 1, one might be curious why there are no differences between the groups at the slower periods, especially in the cases where the group 1 signal is so strong. At these particular sequencies, the variance of the transform is relatively large [and hence, so is (3.8)], thus hindering our ability to detect differences, if any, between the group signals.

In summary, the results of the analyses lead us to conclude that “normal” infant sleep-state cycling is composed of a broad range of cycles ranging from slower periods of approximately 10, 15, 20, and 25 minutes to faster periods of approximately 1–5 minutes. Moreover, our results indicate that there is an effect of maternal alcohol use (at moderate levels) on the sleep-state cycling of infants (we also found a maternal alcohol effect on other infant sleep

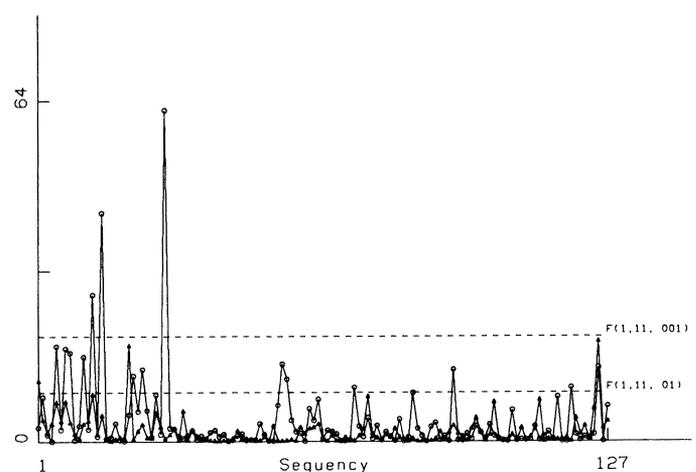


Figure 4. F Statistic (3.5) for Testing for a Common Signal in Group 1 (○) and Group 2 (△).

Table 1. Summary of the Values of the Test Statistics (3.5) and (3.9) at Selected Sequencies

Index (m)	Sequency (switches per minute)	Period (minute)	Common signal F statistic (3.5), group 1	Common signal F statistic (3.5), group 2	Comparison between groups, F statistic (3.9)
1	.008	128.00	2.52	11.23 ^b	.10
5	.039	25.60	17.83 ^b	7.23 ^a	2.18
7	.055	18.29	17.38 ^b	7.37 ^a	1.07
8	.063	16.00	16.56 ^b	3.45	2.61
11	.086	11.64	15.80 ^b	3.20	.22
13	.102	9.85	27.52 ^c	8.71 ^a	3.58
15	.117	8.53	42.80 ^c	4.75	2.76
21	.164	6.10	3.96	16.18 ^b	.16
22	.172	5.82	12.23 ^b	.33	1.84
24	.188	5.33	13.46 ^b	3.10	3.43
25	.195	5.12	5.73 ^a	.63	5.44 ^a
29	.227	4.41	62.14 ^c	.44	7.05 ^b
55	.430	2.33	14.45 ^b	.00	9.69 ^b
71	.555	1.80	9.83 ^b	.03	3.08
93	.727	1.38	13.42 ^b	2.92	8.84 ^b
119	.930	1.08	10.08 ^b	.00	5.32 ^a
124	.969	1.03	1.32	6.00 ^a	5.98 ^a
125	.977	1.02	8.30 ^a	12.44 ^b	.22

^a Exceeds .05 threshold.
^b Exceeds .01 threshold.
^c Exceeds .001 threshold.

outcome variables recorded in this study; see Scher et al. (1988). Differences between ultradian sleep cycles in the two groups may reflect differences in central nervous system maturation. The disturbances in the sleep-state cycling of the exposed group suggests an alteration in the development and expression of diverse neurotransmitter sections within the brain stem and forebrain connections that are associated with the expression of active and quiet sleep. Nevertheless, the small number of replicates in each sample makes it imperative that more infant sleep studies be performed before any definitive conclusions about the effects of moderate maternal alcohol consumption on infant sleep cycling are drawn. We are now collecting more sleep-state records.

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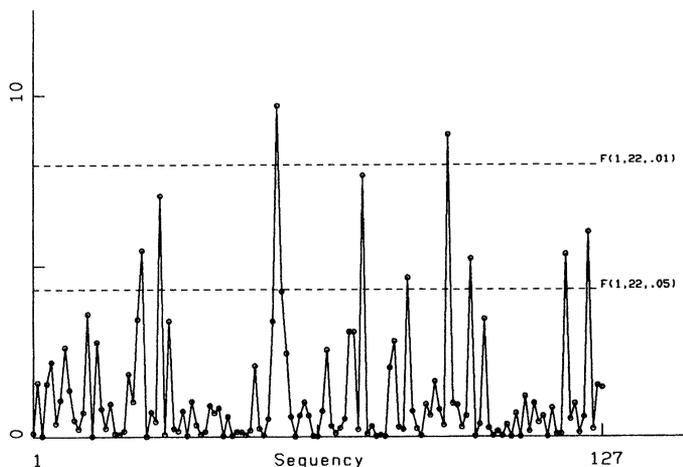


Figure 5. F Statistic (3.9) for Comparing the Signals of Group 1 and Group 2.

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